In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 14-1187V
Filed: June 25, 2021
PUBLISHED

ROBERT MADIGAN,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

Special Master Horner

Influenza Vaccine; Sudden Sensorineural Hearing Loss; SSNHL; Stress Response Theory

Andrew Downing, Van Cott & Talamante PLLC, Phoenix, AZ, for petitioner.
Christine Mary Becer, U.S. Department of Justice, Washington, DC, for respondent.

RULING ON ENTITLEMENT[1]

On December 10, 2014, petitioner filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10-34 (2012),[2] alleging that the influenza vaccine that petitioner received on December 9, 2011, caused him to suffer unilateral sudden sensorineural hearing loss ("SSNHL") in his left ear. (Pet., p. 1.) For the reasons set forth below, I conclude that petitioner is entitled to compensation for his SSNHL.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations, including showing that an individual received a vaccination covered by the statute;

[1] Because this decision contains a reasoned explanation for the special master's action in this case, it will be posted on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002. *See* 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). **This means the decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), petitioner has 14 days to identify and move to redact medical or other information the disclosure of which would constitute an unwarranted invasion of privacy. If the special master, upon review, agrees that the identified material fits within this definition, it will be redacted from public access.

[2] Within this decision, all citations to § 300aa will be the relevant sections of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

received it in the United States; suffered a serious, long-standing injury; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a *causal link* between the vaccination and the injury. In some cases, the petitioner may simply demonstrate the occurrence of what has been called a "Table Injury." That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the "Vaccine Injury Table," corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. If so, the Table Injury is presumed to have been caused by the vaccination, and the petitioner is automatically entitled to compensation, unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. § 300aa-13(a)(1)(A); § 300 aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B).

In many cases, however, the vaccine recipient may have suffered an injury *not* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient's injury was "caused-in-fact" by the vaccination in question. § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In such a situation, of course, the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991). In this case, petitioner alleges that he suffered SSNHL, which is not listed on the Vaccine Injury Table relative to any vaccine. Accordingly, petitioner must satisfy this burden of proof for a cause-in-fact claim.

The showing of "causation-in-fact" must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); *see also Althen*, 418 F.3d at 1279; *Hines*, 940 F.2d at 1525. Under that standard, the petitioner must show that it is "more probable than not" that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. The petitioner need not show that the vaccination was the sole cause of the injury or condition, but must demonstrate that the vaccination was at least a "substantial factor" in causing the condition, and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, the petitioner must supply "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury;" the logical sequence must be supported by "reputable medical or scientific explanation, *i.e.*, evidence in the form of scientific studies or expert medical testimony." *Althen*, 418 F.3d at 1278; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. § 300aa-13(a)(1).

In what has become the predominant framing of this burden of proof, the *Althen* court described the "causation-in-fact" standard, as follows:

> Concisely stated, Althen's burden is to show by preponderant evidence that the vaccination brought about her injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury. If Althen satisfies this burden, she is entitled to recover unless the [government] shows, also by a preponderance of the evidence, that the injury was in fact caused by factors unrelated to the vaccine.

Althen, 418 F.3d at 1278 (citations omitted). The *Althen* court noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. *Id*. at 1279-80. That expert's opinion must be "sound and reliable." *Boatmon v. Sec'y of Health & Human Servs*., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). The *Althen* court also indicated, however, that a Program fact-finder may rely upon "circumstantial evidence," which the court found to be consistent with the "system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants." 481 F.3d at 1280.

II. Procedural History

Petitioner filed this claim on December 10, 2014, alleging both SSNHL and cognitive impairment. (Pet.) This case was first assigned to Special Master Millman. (ECF No. 4.) Petitioner subsequently filed medical records to support his claim. (ECF Nos. 11-20.) An initial status conference was held on February 5, 2015. Special Master Millman indicated that if petitioner intended to pursue allegations of cognitive impairment, petitioner would need to file neurology records. (ECF No. 24.) In a subsequent status conference, petitioner clarified that he "will not move forward with his allegation of cognitive impairment." (ECF No. 25.) Following a substitution of counsel, petitioner filed additional medical records and a Statement of Completion. (ECF Nos. 37, 39.)

Petitioner initially filed an expert report from one of his treating physicians, Barry S. Erner, D.O. (ECF No. 46; Ex. 14.) Respondent filed his Rule 4 report, recommending against compensation and an expert report from infectious disease specialist Kenneth H. Fife, M.D., Ph.D. (ECF Nos. 52-53.) Following a motion to withdraw by petitioner's second counsel, petitioner proceeded *pro se* for over six months until Mr. Andrew Downing was substituted as counsel of record on June 28, 2018. (ECF Nos. 72, 78.) Petitioner then filed an expert report from otolaryngologist, George W. Hicks, M.D. (ECF No. 80; Ex. 50.) Supplemental expert reports by Drs. Fife and Hicks were subsequently filed. (ECF Nos. 81, 82, 86.)

On March 4, 2019, Special Master Millman issued an order stating that, "[w]hen this case is transferred to another special master upon [her] retirement, the new special master will schedule a hearing date." (ECF No. 87.) This case was reassigned to my

docket on June 7, 2019. (ECF No. 89.) On December 30, 2019, the parties filed a joint status report, confirming that this case was ripe for an entitlement hearing. (ECF No. 91.) A two-day entitlement hearing was scheduled to commence on April 20, 2020; however, it was postponed due to the Covid-19 pandemic. (ECF Nos. 92; Scheduling Order (Non-PDF), 3/31/2020.)

A two-day entitlement hearing was ultimately held on January 28 and 29, 2021. (*See* ECF Nos. 119-20, Transcript of Proceedings ("Tr"), January 28 and 29, 2021). Petitioner and his wife, Teresa Madigan, both testified. Additionally, petitioner elicited testimony from petitioner's primary care physician, Dr. Nicora, and his otolaryngology expert, Dr. Hicks. Respondent presented expert testimony from Dr. Fife.

This case is now ripe for resolution of entitlement.

III. Factual History[3]

a. Medical Records

i. Pre-Vaccination Records

Prior to December 15, 2011, petitioner did not complain about hearing loss to his primary care physician, Dr. Michael S. Wein. (Ex. 1, p. 2.) Petitioner had a colonoscopy in December 2010 and had four polyps removed. (*Id*. at 15, 37.) Petitioner's previous medical history included a five-day headache of unclear etiology, elevated levels of glucose and cholesterol, low levels of MPV, and hyperlipidemia. (*Id.* at 23-27, 41-50.)

On January 17, 2011, petitioner had a follow-up visit with Dr. Wein regarding hypertension and hypercholesterolemia. (*Id*. at 6.) Petitioner denied having any side effects to taking Lipitor and his blood pressure was elevated at this visit. (*Id*.) Dr. Wein started petitioner on lisinopril.

At petitioner's next follow-up visit on February 17, 2011 with Dr. Wein, petitioner's "blood pressure has improved after starting lisinopril." (*Id*. at 8.) On March 16, 2011,

[3] Petitioner's prior counsel initially filed his first ten exhibits marked as Exhibits A through J. Special Master Millman struck those exhibits and directed petitioner to use numbered exhibits. (ECF No. 10.) Petitioner subsequently refiled the exhibits, redesignating the exhibits as Exhibits 1-10 in the docket text, but failing to correct the designations and bates stamping within the documents themselves. (ECF Nos. 11-20.) On January 20, 2021, current counsel refiled Exhibits 1-10 in order to add correct bates stamping. However, counsel newly designated these documents as Exhibits 90-99. Accordingly, Exhibits 1-10 and 90-99 are duplicates. However, prior counsel also used cover sheets and included those cover sheets in the bates numbering (i.e. the first substantive page is marked as page 2). In filing Exhibits 90-99, current counsel did not include exhibit cover sheets. Accordingly, the page numbering among the two sets of exhibits does not align despite the exhibits being duplicates. At the same time, petitioner refiled petitioner's initial affidavit as Exhibit 89, noting that it had previously been filed without any exhibit designation. This decision cites to the medical records as designated by filing at ECF Nos. 11-20 as Exhibits 1-10. Page citations are to the bates numbering notwithstanding that the bates stamping incorrectly identifies the exhibits by letter. Petitioner's affidavit is cited as Exhibit 89.

petitioner saw Dr. Wein with a complaint of pain which occurred after tripping over his dog and falling onto his wrist. (Ex. 1, p. 13.) Petitioner had pain and swelling on the dorsum of his wrist at this visit, but no pain in his hand or elbow. (*Id*.) Dr. Wein prescribed petitioner a lace up wrist brace and recommended ice and NSAIDs. (*Id*.) Petitioner saw Dr. Wein for an annual physical on June 17, 2011. (*Id*. at 15.) Petitioner complained of some osteoarthritic changes in his hands, but his physical examination was normal. (*Id*. at 16.) He received a Zostavax vaccination at his visit. (*Id*. at 15.) Petitioner's laboratory results were unremarkable. (Ex.1, pp. 18-21.)

Prior to vaccination, petitioner raised the issue of stress multiple times with Dr. Wein. (*Id.* at 6, 28.) During his 2010 annual exam on April 23, 2010, petitioner indicated that he had recently undergone a period of financial strain that had caused him to lose insurance and delay medical care. (*Id*. at 28.) He indicated that his stress had reduced by that time; however, he again raised the issue of stress on January 17, 2011, in connection with a follow-up regarding hypertension and hypercholesterolemia. (*Id*. at 6.) He attributed his stress to managing his mother's estate.[4] (*Id*.) Shortly thereafter, petitioner began to suspect he had low testosterone, though he had no "significant symptoms or concerns." (*Id*. at 8.) Subsequent testing revealed borderline low testosterone and petitioner was referred to Dr. Rudin, an endocrinologist. (Ex. 1, p. 12.) Upon review of repeat testing showing normal testosterone levels, Dr. Rudin recommended against testosterone therapy. (Ex. 49, p. 180.) Subsequently, however, on September 25, 2011, petitioner reported to Barry Erner, D.O. that he was experiencing "reduced energy [and] sexual interest, can't seem to loose [*sic*] weight, general malaise." (*Id*. at 84.) Petitioner saw Dr. Erner on November 7, 2011 for low back pain, myalgia, and muscle spasm. (*Id*. at 80-81.) During 2011 Dr. Erner prescribed both testosterone and Valium. (*Id*. at 169, 172.)

ii. Vaccination and Initial Treatment

On December 9, 2011, petitioner visited Dr. Wein for scalp lesions. (Ex. 2, p. 2.) Petitioner also noticed "slightly swollen lymph nodes in the back of his neck on the right side." (*Id*.) Dr. Wein suspected "this is a reactive lymph node secondary to folliculitis on the scalp." (*Id*.) Petitioner was prescribed doxycycline and received a flu vaccination. (*Id.* at 2; Ex. 1, p. 3.)

Petitioner was seen at Northern Westchester Hospital on December 13, 2011, for complaints of vomiting, ringing in left ear, and tingling in arms that started a day earlier. (Ex. 3, p. 4.) Petitioner reported receiving the flu short three days prior to being admitted and that he started doxycycline two days prior to admission for an infected lymph node. (*Id*.) Petitioner felt better after receiving "IV hydration but still nauseated – decreased hearing in left ear, feeling hazy in head and slightly unsteady." (*Id*. at 6.) Petitioner's laboratory results revealed high levels of neutrophils but low levels of lymphocytes. (*Id*. at 22.) Petitioner's radiology showed no evidence of acute pulmonary

[4] The exact notation in the medical record is "He does state that he has been under a lot of stress recently handling his mother's state [*sic*]." (Ex. 1, p. 6.) Interestingly, petitioner was doing work for his mother's estate around the time of onset of his SSNHL. (Tr. 40-41.)

disease. (Ex. 4, p. 4.) Petitioner was discharged with labyrinthine disorder. (Ex. 3, p. 6.)

Almost a week later after seeing Dr. Wein for scalp lesions on December 15, 2011, petitioner returned to Dr. Wein for a "follow-up from emergency department." (Ex. 2, p. 3.) Petitioner reported taking doxycycline for three days and "[s]ubsequently, the lymph node completely resolved and the lesions on his scalp improved considerably." (*Id*.) Petitioner was treated with Zofran, Antivert, and IV fluids at the emergency room and reported to Dr. Wein that he felt "completely back to normal with the exception of decreased hearing in the left ear." (*Id*.) Dr. Wein was concerned about the possibility of Meniere's disease or a viral vestibulitis but felt it was not likely a reaction to the doxycycline. (*Id*.)

On the same day, petitioner saw Dr. William D. Losquadro for a consultation referred by Dr. Wein. (Ex. 4, p. 2.) Petitioner complained of sudden hearing loss in the left ear and started hearing static-like sounds, although his right ear felt fine. (*Id*.) Dr. Losquadro noted, upon physical examination, "bilateral tympanic membranes dull in appearance." (*Id*.) Dr. Losquadro assessed petitioner with sudden sensorineural hearing loss on the left and recommended a 10-day course of steroids and Valacyclovir. (*Id*.)

Petitioner visited another ENT specialist, Dr. Tali Lando, on December 16, 2011. (Ex. 5, p. 2; Ex. 12, p. 13.) Petitioner reported "ringing in both ears with violent nausea and vomiting throughout the day with imbalance and vertigo," and had aural fullness in his left ear. (Ex. 5, p. 2.) Additionally, "[a]bout a decade ago, he did have an episode of vertigo lasting for a few minutes and was found to have very high blood pressure but was self-resolved." (*Id*.) Dr. Lando indicated that due to petitioner's young age, Weber and Rinne's tests could not be performed; however, an audiogram showed "absent left reflexes, left severe profound hearing loss." (*Id*.) Dr. Lando assessed petitioner with labyrinthitis of left ear and sensorineural hearing loss. (*Id*.) On the same day, petitioner underwent a brain MRI, which showed "no intracranial hemorrhage or acute infraction. No cerebellopontine angle mass." (Ex. 6, p.2.)

On December 22, 2011, petitioner saw ENT specialist Katrina R. Stidham, M.D., for follow-up and for consideration of inner ear perfusion of steroids. (Ex. 5, p. 5; Ex. 12, p. 15.) Petitioner did not have any significant improvement in hearing loss. (Ex. 5, p. 5.) Petitioner's physical examination was normal. Petitioner was assessed with sensorineural hearing loss, labyrinthitis of left ear, vertiginous syndromes and other disorders of vestibular system. (*Id*.) Dr. Stidham indicated that petitioner was a "good candidate for inner ear perfusion." (*Id*.) In a follow-up visit with Dr. Stidham on December 28, 2011, petitioner reported having pain for about one hour after his last injection. (Ex. 5, p. 7; Ex. 12, p. 17.) Petitioner had another steroid inner ear perfusion. (Ex. 5, p. 8.) Petitioner's December 28, 2011 audiogram indicated "pure tone thresholds unchanged from last audio." (*Id*. at 9.)

Petitioner returned to Dr. Stidham on January 12, 2012 for a follow-up visit relating to his labyrinthitis. (Ex. 5, p. 10; Ex. 12, p. 19.) Petitioner reported "doing ok, but still feeling some problems with balance, stiffness and nausea. Also notes no subjective improvement in hearing." (Ex. 5, p. 10.) Dr. Stidham also concluded there had been no improvement in hearing. (*Id*.) Petitioner's audiogram noted "moderately-severe to profound" sensorineural hearing loss with a slight improvement from previous audiograms. Continued monitoring was recommended. (*Id*. at 12.) Petitioner continued visiting Dr. Stidham for follow-up appointments thereafter through January and February of 2012. (Ex. 12, pp. 21-24.)

Also, on January 12, 2012, petitioner visited Balance Center Westchester Medical Center and was examined by Amanda Muldoon, Au.D. (Ex. 5, p. 17; Ex. 12, p. 2.) Petitioner's sensory organization test, rotational vestibular test, and videonystagmography[5] were within normal limits. (Ex. 5, pp. 17-18.) Petitioner's "Basic Balance Function test results indicate vestibular responses to bithermal air calorie stimulation to be within normal limits in both ears on the VNG," and petitioner's rotational trapezoidal step test may indicate migraine related dizziness or motion intolerance. (*Id*. at 18.)

On January 23, 2012, petitioner visited Dr. Barry S. Erner, with a complaint of left ear feeling fuller. (Ex. 49, p. 78.) Dr. Erner noted viral syndrome or vaccine reaction as a possible diagnosis. (*Id*. at 79.)

iii. Subsequent Treatment Records

Petitioner had a psychiatric evaluation on July 23, 2012 with Mark Weinberger, Ph.D. (Ex. 86.) Petitioner reported problems with his sleeping and feeling overwhelmed over having sudden onset of hearing loss. (*Id*. at 1.) Petitioner also reported "significant depressive symptoms related to his hearing loss." (*Id*. at 2.) Additionally, petitioner complained of short-term memory loss and "often using the wrong words," where petitioner attributed his cognitive deficits to be related to his hearing loss. (*Id*.) Dr. Weinberger diagnosed petitioner with adjustment disorder with depressed mood and cognitive disorder and recommended individual psychological therapy. (*Id*. at 4.) Dr. Weinberger indicated that petitioner's prognosis was fair. (*Id*.)

Petitioner had an initial visit and examination with Dr. Pamela C. Roehm on April 24, 2012 for a second opinion regarding petitioner's profound sudden hearing loss in his left ear. (Ex. 7, pp. 8-10; Ex. 13, pp. 9-11.) Petitioner returned to Dr. Roehm on September 25, 2012 "following up with complaints of left nonpulsatile tinnitus following a left sudden sensorineural hearing loss." (Ex. 7, p. 2; Ex. 13, p. 2.) Dr. Roehm noted that petitioner's "ENG[6] (performed after his sudden hearing loss) showed no anomalies

[5] This test, abbreviated "VNG," measures a type of involuntary eye movement called nystagmus. The test screens for vestibular disorders. *See* https://medlineplus.gov/lab-tests/videonystagmography-vng/, last accessed June 21, 2021.

[6] "ENG" refers to electronystagmography. This test is similar to the VNG test, but uses electrodes to measure eye movement instead of video. *See* https://medlineplus.gov/ency/article/003448.htm, last

other than those consistent with intolerance/migraine." (Ex. 7, p. 2) Petitioner's wife was concerned with petitioner's memory because since his hearing loss, "he has had difficulty multitasking, frequently forgets ongoing tasks, and substitutes words for more commonly used ones." (*Id*. at 2-3.) Dr. Roehm listed tinnitus of left ear as petitioner's primary diagnosis and sudden idiopathic hearing loss of left ear as well. (*Id*. at 4.) Regarding petitioner's hearing loss, Dr. Roehm indicated that "conventional hearing aid will not be helpful given the poor left discrimination, we will put him on the list for the CI[7] trial." (*Id*.) Dr. Roehm indicated that petitioner's memory issues may be related to his sleep deprivation. (*Id*.)

Petitioner saw Dr. Erner again on March 5, 2013, and occasionally every several months from 2013 through 2017. (*See generally* Ex. 49.) Petitioner continued suffering from left ear hearing loss. (*Id*. at 1-80.) Petitioner was seeing Dr. Erner for chronic pain and continued having his prescriptions refilled. (*Id*. at 90-119.)

On March 11, 2013, petitioner saw Dr. John C. Scott for audiometric examination and follow up regarding his sudden sensorineural hearing loss and cognitive function. (Ex. 8, p. 2.) Petitioner's physical examination was normal and Dr. Scott recommended a repeat hearing test on a yearly basis. (*Id*. at 2-3.) Petitioner was not interested in CROS hearing aid placement. (*Id*. at 3.)

Petitioner had a consultation regarding his sudden sensorineural hearing loss with Dr. Frank Lin at Johns Hopkins on April 23, 2013. (Ex. 10.) Petitioner reported that he had "persistent inability to localize sound, problems with sounds discrimination, as well as what he believes are subtle affects [*sic*] on his cognitive functioning." (*Id*. at 2.) Petitioner presented his neurocognitive exam to Dr. Lin, who noted that petitioner's neuropsychologist was not able to discern any obvious cognitive problems. Petitioner was also seen by a psychiatrist who attributed petitioner's symptoms at least partly to ADHD and anxiety. (*Id*.) Dr. Lin opined that petitioner did not have any "appreciable recovery" of his hearing loss and petitioner's testing "demonstrated no obvious evidence of any more insidious causes for his sudden hearing loss." (*Id*. at 3.) Dr. Lin thought it was reasonable to start petitioner with a CROS hearing aid to help with petitioner's symptoms. Additionally, Dr. Lin opined that petitioner's tinnitus could be exacerbated by his single sided deafness and increased stress. (*Id*.) Petitioner's audiologic evaluation in conjunction with his appointment with Dr. Lin indicated that his "pure tone results revealed normal hearing sensitivity in the right ear, [but] the left ear results revealed a moderately-severe to severe sensorineural hearing loss." (*Id*. at 5.)

On June 21, 2013, petitioner underwent a comprehensive functional vocational capacities evaluation and employability analysis for his Social Security disability claim. (Ex. 87.) Andrew J. Pasternak, the vocational expert, indicated that petitioner's

---

accessed on June 21, 2021. This is likely a reference to the above-referenced VNG test of January 12, 2012.

[7] "CI" likely stands for cochlear implant. The records appear to indicate that petitioner expressed interest in a cochlear implant for singled sided deafness. (Ex. 7, p. 4.)

"conditions are attributed apparently to a virus that has resulted in hearing loss that began to affect him beginning in 12/13/2011 and gradually became worse with on-going tinnitus." (*Id*. at 3.) Petitioner worked in graphic design and expressed an interest in gardening, but could not continue classes due to his hearing issues. (*Id*. at 4.) In Mr. Pasternak's opinion, petitioner was "functionally incapable of performing the duties of his former positions as both a Property Manager and an Art Director/Graphic Designer on a sustained full-time, regular competitive basis. Nor does he have the capacity to achieve a competitive level for any other jobs in the local or national economy." (*Id*. at 6.)

Petitioner's November 19, 2014 audiogram, which was similar to his March 13, 2013 audiogram, showed that petitioner's left ear had "a potentially flat moderately severe-to-severe sensorineural hearing loss with discrimination scores of only 8%." (Ex. 10, p. 3.) Dr. Lin indicated to petitioner that "his clinical history and audiologic findings are consistent with an idiopathic or possibly a viral left-sided sudden sensorineural hearing loss." (*Id*.)

Starting in 2018, petitioner saw Dr. Giovanni Angelino[8] for pain management of his lumbar radiculopathy at Northern Westchester Hospital. (Ex. 85.) Dr. Angelino performed multiple lumbar steroid injection with x-ray and sedation on petitioner. (*Id*., *e.g.*, at 33-43, 69-72, 105.) Most recently, petitioner was admitted to Northern Westchester Hospital for a radio frequency ablation lumbar medial branch nerve facet injection with x-ray and sedation on October 1, 2019. (*Id.* at 1-11.) Additionally, petitioner had a small excisional biopsy on his upper lip lesion in November 26, 2019. (Ex. 83.)

Petitioner underwent therapy sessions in 2017 through 2020 with Robert Muller, Ph.D. (Ex. 84.) Dr. Muller provided a clinical summary update on February 22, 2020. Petitioner reported difficulty getting pleasure from activities that he had previously enjoyed due to his problems hearing and comprehending others. Dr. Muller reported that, "[a]s a result, [petitioner] tends to avoid social interactions. This general withdrawal has resulted in social isolation which exacerbates feelings of loneliness and hopelessness." (*Id*. at 1.) Additionally, Dr. Muller indicated that due to petitioner's sensory and cognitive deficits, petitioner had a difficulty maintaining meaningful employment. (*Id*.)

b. Barry S. Erner, D.O., Report

One of petitioner's treating physician, Barry S. Erner, provided a written opinion supporting petitioner's theory of vaccine-causation. Dr. Erner received his Doctor of Osteopathic Medicine from New York College of Osteopathic Medicine and is certified in

[8] Petitioner previously saw Dr. Angelino for low back, left buttock and thigh pain in 2013 and received lumbar epidural steroid injections then as well. (Ex. 85, pp. 151-212.)

medical acupuncture. Dr. Erner previously taught at his alma mater and has been in private practice since 1988. (ECF No. 65-2.)[9]

As explained above, petitioner first consulted Dr. Erner on September 26, 2011, "with chief complaints of reduced energy, decreased sexual interest, difficulty losing weight, and general malaise." (Ex. 14, p. 1; *see also* Ex. 49, p. 84.) Dr. Erner repeated laboratory testing for the evaluation of hypothyroidism and hypogonadism and the results were unremarkable. (*Id*.) Petitioner sought further treatment from Dr. Erner on November 7, 2011, after slipping and experiencing lower back pain and myalgias of both the cervical and lumbar paravertebral musculature. (*Id*.) Dr. Erner treated petitioner again on January 23, 2012, post vaccination, when petitioner complained of left ear sensorineural hearing loss (SNHL). (*Id*.) Petitioner told Dr. Erner that within 72 hours of receiving his influenza vaccination, petitioner experienced a new set of symptoms from his mild infection, including "vomiting, disorientation, fullness in the left ear, and loss of hearing in the left ear." (*Id*.) Dr. Erner indicated that since then, petitioner "has sought and received treatment for Labyrinthitis, chronic pain in cervical and lumbar regions, insomnia, mood disorder, and loss of hearing in the left ear." (*Id*.)

Dr. Erner opined that "there is a clear, temporal, and causative relationship between the Influenza vaccination and [petitioner's] hearing loss." He explained that vaccines have previously been reported as a cause of hearing loss, citing two case reports involving Hepatitis B vaccinations.[10] However, he further stated that the "Influenza vaccine has been implicated as causing a mild but clearly measurable acute phase response with an increase in inflammatory cytokines and acute phase proteins," and additionally, that "[v]iral infections are a known causative agent in both congenital hearing loss and in the induction of host immune mediated nerve damage." (Ex. 14, p. 2.)

Regarding petitioner's case, Dr. Erner opined that petitioner "was suffering from an infection and then received the Influenza vaccine, which, when superimposed on his condition, caused a stress and inflammatory response which disturbed and damaged his auditory function." (*Id*.) Dr. Erner concluded that the "infectious underling pathology and an augmented immune response from the stress inflammatory response from the vaccination is likely the sequence of events that led to [petitioner's] left ear SNHL."[11] (*Id*.)

---

[9] Dr. Erner's CV was filed as Ex. 50 from prior counsel. There is another Ex. 50, which is an article filed by current counsel, Mr. Downing.

[10] Biacabe et al., *A Case Report of Fluctant Sensorineural Hearing Loss After Hepatitis B Vaccination*, 24 AURIS NASUS LARYNX 357 (1997) (Ex. 18); Orlando et al., *Sudden Hearing Loss in Childhood Consequent to Hepatitis B Vaccination: A Case Report*, 830 ANNALS OF NY ACADEMY OF SCIENCES 319 (1997) (Ex. 47).

[11] Because petitioner subsequently retained an expert with qualifications in otolaryngology who testified in this case, Dr. Erner's opinion will not be extensively discussed. However, Dr. Ener also cited a substantial number of articles in his report. Some of the literature discussed in the analysis below was initially filed by petitioner in connection with Dr. Erner's report.

c. Christopher Nicora, M.D., Letter and Testimony

On November 19, 2014, petitioner's primary care physician, Dr. Nicora, wrote a "to whom it may concern" letter that was initially filed in this case as Exhibit 9. That letter indicated that onset of petitioner's SSNHL was three days following his 2011 flu vaccination and that "[t]he timing of his hearing loss points to the influenza vaccine being the probable cause." (Ex. 9, p. 1.) Dr. Nicora also subsequently testified at the entitlement hearing. (Tr. 18-34.)

Dr. Nicora is a doctor of internal medicine and petitioner's primary care provider. (Tr. 19-20.) His curriculum vitae is filed as Exhibit 100. He began seeing petitioner after onset of his SSNHL in April of 2012. (Tr. 20-21.) In treating petitioner in the aftermath of his hearing loss, Dr. Nicora gave consideration to whether petitioner's prior history suggested his hearing loss was related to either his flu vaccine or the doxycycline he was taking at the same time. (Tr. 22-23.) After conducting research, Dr. Nicora felt comfortable ruling doxycycline out as being non-ototoxic. (Tr. 23.) Instead, Dr. Nicora felt the systemic inflammatory effects of the flu vaccine were a more likely cause of SSNHL in petitioner's case. (Tr. 23-24, 30.) As part of petitioner's ongoing care, Dr. Nicora recommends that petitioner avoid future flu vaccinations. (Tr. 24.)

d. Petitioner's Testimony

In the affidavit filed with his petition, petitioner averred that he "remained in relatively good health with no problems related to hearing until [he] was diagnosed with sudden unilateral sensorineural hearing loss to the left ear on or about December 15, 2011, six days after receiving the [flu] vaccine on December 9, 2011." (Ex. 89, p. 1.) Petitioner started experiencing symptoms of nausea, dizziness, ringing sounds, vomiting, diarrhea, distortion of sound, vertigo, and inability to walk when he woke up on December 12, 2011. (*Id*. at 2.) He continued to experience such symptoms and eventually was admitted to Northern Westchester Hospital emergency room the next day. Petitioner could not hear anything out of his left ear by December 14, 2011. (*Id*.) Petitioner was told that his condition is permanent and there has been no change in his hearing loss. (*Id*. at 4.) During the entitlement hearing petitioner provided additional testimony generally consistent with these recollections. (Tr. 34-72.)

e. Mrs. Madigan's Testimony

Petitioner's wife, Teresa Madigan, submitted a letter dated November 8, 2012, detailing the events surrounding petitioner's unilateral sensorineural hearing loss in his left ear. (Ex. 82.) On December 9, 2011, petitioner called his wife to let her know that he had gotten the flu shot and that Dr. Wein prescribed him antibiotics for his swollen lymph glands. Mrs. Madigan stated that petitioner walked the dog and made dinner that night. About three days later, Mrs. Madigan came home and found petitioner on the couch feeling unwell. Petitioner proceeded to vomit, and Ms. Madigan called her sister, who was a nurse for advice. The next day, Mrs. Madigan called Dr. Wein's office to schedule an appointment, but petitioner insisted on going to the hospital instead. (*Id*. at

1-2.) Petitioner was discharged on the same day and slept as soon as he got home. Mrs. Madigan stated that the next morning, petitioner woke up and told her he couldn't hear anything out of his left ear. Petitioner then proceeded to vomit again and complained about his hearing. Mrs. Madigan stated that petitioner never regained his hearing. Mrs. Madigan also offered similar testimony during the entitlement hearing. (Tr. 5-16.)

IV. Summary of Experts' Opinions

a. Petitioner's Expert: George W. Hicks, M.D.

Petitioner additionally relied on the expert opinion of George W. Hicks, M.D., to support his claim. Dr. Hicks is board certified in otolaryngology (head and neck surgery) and currently holds a teaching position at Indiana University School of Medicine and Marian University School of Medicine. Additionally, Dr. Hicks is an otologist/neurotologist at Veterans Administration Hospital Indianapolis and board member of Hear Indiana. (Ex. 51.) He is in private practice providing consultation for ear, hearing, and balance disorders.

Dr. Hicks explained that sudden sensorineural hearing loss (SSNHL) is "a 30 [decibel ("dB")] decrease in hearing nerve function over three contiguous audiometric frequencies occurring within three days or less," and that "[i]t does not occur in both ears as with a systemic inflammation, but unilaterally due to selective damage of the inner ear cochlea of only one ear." (Ex. 50, p. 2.) Dr. Hicks explained that only 15% of SSNHL cases have known causes while 85% are considered idiopathic (ISSNHL), meaning of unknown etiology. (*Id*.) According to Dr. Hicks, SSNHL can be reversible if the patient is seen within 14 days or less of onset and "the diagnosis of SSNHL is based on a careful well-documented history, a neurotologic examination, especially of the cranial nerves, an ear exam using pneumatic otoscopy or microscopic examination, audiometry supported by tuning fork tests, and an MRI for all patients." (*Id*.) In contrast, "[l]aboratory tests are, in most cases, not beneficial." (*Id*.) Additionally, Dr. Hicks noted that, "[i]nitial symptoms of SSNHL may present with symptoms such as aural fullness and muffled hearing, symptoms which may be erroneously attributed to cerumen, congestion, or bacterial infection (otitis media)." (*Id*. at 3.) Dr. Hicks stressed that "there are significant limitations in evaluating inner ear disease, most importantly is the inaccessibility of the intracochlear tissue during lifetime." (*Id*. at 6.)

Dr. Hicks opined that petitioner has suffered SSNHL that has led to permanent hearing loss. (Tr. 95; Ex. 50, pp. 6, 9.) Dr. Hicks also highlighted the opinions of petitioner's treating physicians, Drs. Erner and Nicora, that identified petitioner's vaccination as a probable cause to petitioner's symptoms and hearing loss. (Ex. 50, p. 11.) Dr. Hicks acknowledged that petitioner's diagnosis from his treating physicians was idiopathic SSNHL; however, he explained that there are common theories of causation for idiopathic cases, including "vascular occlusion or ischemia, intra-labyrinthine membrane rupture, immunologic autoimmune inner ear disease, viral infection, and the stress response theory." (*Id*. at 6.) Dr. Hicks ruled out vascular

occlusion or ischemia and intra-labyrinthine membrane break and offered two separate theories, a stress response theory and an immunologic theory, that he opined operated alone or in combination to cause petitioner's SSNHL. (*Id*. at 7-9.)

First, Dr. Hicks proposed that the so called "stress response theory" explains how the flu vaccine could cause damaging inner ear inflammation. That theory, explained in greater detail in the analysis below, posits that "a systemic stressor can increase circulating cytokines, activate NFkB[12] which initiates an inflammatory response in the lateral wall of the cochlear locally, leading to damage of cochlear homeostasis." (*Id*. at 7.) In his report, Dr. Hicks initially appeared to suggest that his opinion was based on the idea that "[t]he influenza vaccine entered the ear via the labyrinthine artery." (*Id*. at 9.) During the hearing, however, he clarified that his opinion is not based on the vaccine components entering the ear, but rather on the "consequences of inflammation" reaching the inner ear. (Tr. 153.) He was explicit in the hearing in noting that "[i]f you wanted to be able to needle the inner ear and draw out some viral particles, you are not going to find them." (*Id*.) Dr. Hicks opined that petitioner's "laboratory results from North Westchester Hospital demonstrated elevated neutrophils and may be a contributing factor to an increase in pathologic activation of NFkB along with a storm-like release of inflammatory cytokines due [to] his memory cell immune response from his vaccination."[13] (Ex. 50, p. 7.) Dr. Hicks also noted that "[t]he lack of benefit from anti-viral medication fits the Stress Theory."[14] (*Id*.)

Second, Dr. Hicks suggested that "ISSHL can be explained by a reactivation of a latent virus in a seropositive patient." (*Id*. at 6.) Dr. Hicks then concluded in this context that "ISSNHL is not due to a true viral labyrinthitis, but rather an immunologic or local, unilateral stress-induced response in the inner ear, e.g., reactivated latent HSV-1 within the spiral ganglion." (*Id*.) According to Dr. Hicks, it is well known that HSV-1 latent virus is present in the geniculate ganglion, vestibular ganglion, and the auditory nerve

---

[12] "NF-kB" of "NF-kβ" stands for nuclear factor kappa beta, a transcription factor that is hypothesized to act as a "cellular stress pathway" when abnormally activated by certain cytokines. (*See, e.g*. Merchant, Durband & Adams, *infra*, at Ex. 38, p. 8.)

[13] Dr. Hicks's reference to a "storm-like" increase in cytokines calls to mind the specific concept in immunology of a "cytokine storm." This is generally understood to represent a catastrophic autoimmune reaction. *E.g. Rupert v. Sec'y of Health & Human Servs.,* No. 15-841V, 2021 WL 1832909, at *40 (Fed. Cl. Spec. Mstr. May 1, 2021) (noting that respondent's expert "maintained that 'there is not credible scientific evidence' that vaccines are capable of inducing a cytokine storm" and that "[p]etitioner does not allege that she suffered from a cytokine storm as a result of her vaccine. Cytokine storm is a catastrophic autoimmune response, and there is not evidence of that occurring in Petitioner's case.") In the context of this case, however, given that it is an isolated reference and especially given Dr. Hicks's area of specialty, this reference to a "storm-like" cytokine release is not understood as invoking a "cytokine storm" as discussed in prior cases. Although the stress response theory advanced by Dr. Hicks involves an increase in cytokines, the concept relates specifically to the delicacy of the inner ear and nothing in the literature suggests that the proposed increase in cytokines is catastrophic.

[14] Dr. Hicks initially indicated that petitioner had experienced a double dosing of flu vaccine in 2011. (Ex 50, p. 8.) However, he later retracted that assertion and confirmed that his opinion did not depend on such a double dosing. (Tr. 179.)

spiral ganglion. Latent HSV-1 can be reactivated by innocuous genotoxic stresses or infection and can lead to debilitating symptoms, e.g., Bell's palsy (geniculate), vestibular neuritis (Scarpa's), or SNHL (spiral). (*Id*. at 9.)

Dr. Hicks suggested that "[m]ultiple studies have estimated that latent HSV-1 is present in 60-90 percent of the world's population." (Ex. 52, p. 1.) Due to the inability to safely needle or biopsy the inner ear, "we must opine in terms of probabilities" and therefore, "it is highly likely that [petitioner] had a latent HSV-1 infection which was re-activated by his vaccination leading to otologic consequences." (*Id*.) In addressing the amount of concentration of vaccine sufficient to trigger a cytokine reaction, Dr. Hicks opined that the "fragility and sensitivity of the inner ear to stress of any type is well known to otologists," where "[l]atent HSV-1 can be reactivated from simple genotoxic stress, e.g., excessive sun exposure, fever, the common cold, emotional or physical stress, and other seemingly innocuous events such as a minimal concentration of influenza vaccine." (*Id*.)

In suggesting that the two mechanisms could be combined, Dr. Hicks proposed that the inflammation caused by the flu vaccination spread to petitioner's left inner ear cochlea via hematogenous systemic circulation, which lead to overload as a stress response that reactivated latent HSV-1 in petitioner's spiral ganglion, which then damaged the cochlear homeostasis that caused petitioner's permanent left SSNHL. (Ex. 50, p. 9; Tr. 153.) Dr. Hicks pointed out that petitioner developed severe SNHL within 2-3 days after his flu vaccination and that no more likely cause is available. (*Id*. at 10.) Dr. Hicks stressed that the fact that the inner ear cannot be probed or studied directly means that obtaining direct proof of offered etiologies is unlikely. (*Id*.)

b. Respondent's Expert: Kenneth H. Fife, M.D., Ph.D.

Dr. Kenneth H. Fife is board certified in internal medicine and infectious diseases. (Ex. A, p. 1.) He is currently a professor in the Division of Infectious Diseases with joint appointments in the Departments of Microbiology and Immunology, and Pathology at Indiana University School of Medicine. (*Id*.)

Dr. Fife first responded to Dr. Erner's opinion on respondent's behalf. (Ex. A.) He emphasized that Dr. Erner failed to "present a convincing hypothetical mechanism," having only presented two case reports of children who developed hearing loss following Hepatitis B vaccinations that support mere temporal association and not causation. (*Id*. at 3.) Dr. Fife opined that "petitioner developed idiopathic unilateral sudden sensorineural hearing loss that happened to occur a few days after receipt of the influenza vaccine." (*Id*.) Additionally, Dr. Fife indicated that "none of the professionals who deal with SSHL on a regular basis made any comment in any note to suggest that the vaccine caused the hearing loss." (*Id*. at 2.) Dr. Fife noted that Dr. Nicora's short letter, dated three years post-vaccination, positing petitioner's hearing loss to be vaccine-related did not contain any explanation and that, moreover, Dr. Nicora did not identify himself as an otolaryngologist. (*Id*.)

In response to Dr. Hicks's report, Dr. Fife indicated that because the influenza vaccine is administered intramuscularly in the upper arm and the immunological response takes place locally, "[l]ittle, if any, of the vaccine gets into the venous circulation, and even then very slowly," and "[b]y the time any vaccine mixed with all venous blood in the body and reached the arterial circulation, it would be at miniscule concentrations and would be unable to trigger a cytokine reaction," as Dr. Hicks suggested. (Ex. C, p. 3.) Moreover, "the surface area of the labyrinthine system is irrelevant if there is no antigen in the circulating blood or in the inner ear endolymph." (Ex. E, p. 2.) Dr. Fife further opined that the blood-labyrinthine barrier itself would further stymie entry of molecules into the inner ear. (Ex. H.)

Dr. Fife also criticized Dr. Hicks's theory that the flu immunization activated latent herpes simplex virus type 1 (HSV-1) and triggered an inflammatory response causing petitioner's unilateral hearing loss, especially when there is no evidence from the medical record that petitioner had latent HSV-1. (Ex. C, p. 3.) Dr. Fife further took issue with Dr. Hicks's assertion that petitioner has a latent HSV-1 infection according to statistics. (Ex. E, p. 2.) Dr. Fife stated, "[a]lthough HSV-1 infection is common, the prevalence is falling and the prevalence is influenced by age, socioeconomic status, and other factors. In this case, the chances that the petitioner has latent HSV-1 infection is about 60%, which means there is about a 40% chance that he does not have latent HSV-1." (*Id*.) And in fact, although it is true that an inner ear biopsy is required to prove causation, Dr. Fife opined that "the role HSV-1 [plays] in this process could be investigated with a type-specific HSV-1 antibody test," where a negative test would exclude Dr. Hicks's theory. (*Id*.) During the hearing, Dr. Fife described the science of herpes viral reactivation as "murky." (Tr. 229.) He acknowledged that reactivation has been shown in specific circumstances and has been associated with elevated Interleukin ("IL")-2, but did not agree that the flu vaccine has been shown to reactivate HSV-1. (*Id*.)

Again, Dr. Fife criticized petitioner's reliance on case reports to support causality, especially when "most of the case reports of SSNHL and vaccines in the medical literature implicates vaccines other than influenza vaccine." (Ex. E, p. 3.) Dr. Fife stated that Dr. Hicks "seems to be alone in his opinion that influenza vaccine is a recognized cause of SSNHL." (Ex. E, p. 2.) Dr. Fife listed a recent review of 200 VAERS reports that record only one report related to general hearing loss, and another study of 20 million doses of influenza vaccine administered over a six-year period at Kaiser-Permanente health facilities that found no association between immunization and SSNHL. (*Id*. citing Williams, et al., *Causality assessment of serious neurologic adverse events following 2009 H1N1 vaccination*, 29 VACCINE 8302 (2011) (Ex. D) and Baxter, et al., *Sudden-Onset Sensorineural Hearing Loss after Immunization: A Case-Centered Analysis,* 155(1) OTOLARYNGOLOGY – HEAD AND NECK SURGERY 81 (2016 (Ex. F).) Therefore, Dr. Fife opined that petitioner's case does not meet the criteria for causality because "85% of cases of SSNHL are idiopathic," and here, this "idiopathic case occurred in proximity to an influenza immunization by chance," as "there is no plausible mechanism of causality." (Ex. C, p. 3.)

V. Petitioner has Satisfied the *Althen* Prongs

a. *Althen* Prong One

Under *Althen* prong one, petitioner must provide a "reputable medical theory," demonstrating that the vaccine received can cause the type of injury alleged. *Pafford v. Sec'y of Health & Human Servs.*, 451 F.3d 1352, 1355–56 (Fed. Cir. 2006) (citations omitted). To satisfy this prong, petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994); *Boatmon v. Sec'y of Health & Human Servs*., 941 F.3d 1351, 1359 (Fed. Cir. 2019). However, such a theory must only be "legally probable, not medically or scientifically certain." *Knudsen*, 35 F.3d at 548-49. Petitioner may satisfy the first *Althen* prong without resorting to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1325-26 (Fed. Cir. 2006)).

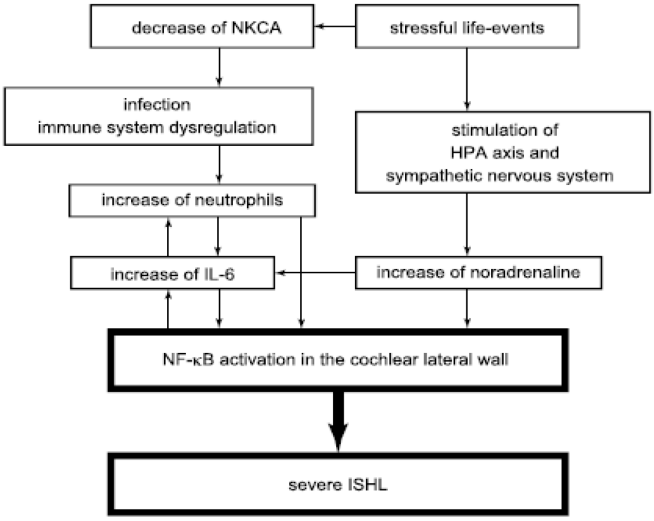
As explained above, sudden hearing loss is defined as rapid onset of hearing impairment of greater than 30 decibels in one or both ears occurring over a 72-hour period. (Robert Stachler, et al., *Clinical Practice Guideline: Sudden Hearing Loss*, 146 OTOLARYNGOLOGY – HEAD AND NECK SURGERY 1 (2012) (Ex. 55, p. 4).) Sensorineural hearing loss is hearing loss related to an abnormality of the cochlea, auditory nerve, or higher aspects of auditory processing. (*Id*.) Between 85-90% of cases of SSNHL remain idiopathic after investigation. (*Id*. at 4-5.) Nonetheless, several etiologies have been proposed, including vascular and viral etiologies. (*Id*. at 4.) The influenza virus, herpes virus, and cytomegalovirus have all been associated with SSNHL. (John Mills and Jacqueline Going, *Review of Environmental Factors Affecting Hearing*, 44 ENVIRONMENTAL HEALTH PERSPECTIVES 119 (1982) (Ex. 15, p. 7).) Reactivation of latent herpes infection within the spiral ganglion has also specifically been proposed as a cause of vestibular dysfunction. (Richard Gacek, Evidence for a Viral Neuropathy in Recurrent Vertigo, 70 ORL 6 (2008) (Ex. 21).) Each proposed etiology is both supported and refuted by the relevant body of medical literature. (Masatsugi Masuda & Jin Kanzaki, *Cause of idiopathic sudden sensorineural hearing loss: The stress response theory*, 3(3) WORLD J. OTORHINOLARYNGOLOGY 42 (2013) (Ex. 23, p. 1).) Some sensorineural hearing loss is considered to be autoimmune; however, that condition is generally bilateral. (C. Arturo Solares, Gordon Hughes, & Vincent Tuohy, *Autoimmune sensorineural hearing loss: an immunologic perspective*, 138 J. OF NEUROIMMUNOLOGY 1 (2003) (Ex. 69).) Dr. Hicks has not opined that petitioner suffered autoimmune sensorineural hearing loss. (Tr. 171.)

More recently, beginning in the 2000's, research spearheaded by Merchant and Adams began to question the suspected causes of SSNHL, especially the theory of a viral etiology. (Saumil Merchant, Marlene Durband & Joe Adams, *Sudden Deafness: Is It Viral?*, 70(1) ORL J. OTORHINOLARYNGOL RELAT SPEC 52 (2008) (Ex. 38) and Saumil Merchant, Joe Adams & Joseph Nadol, Jr., *Pathology and Pathophysiology of Idiopathic*

Sudden Sensorineural Hearing Loss, 26 OTOLOGY & NEUROTOLOGY 151 (2005) (Ex. 59).) These researchers focused on nuclear factor kappa beta ("NF-kβ"), a transcription factor found throughout the body. (Merchant, Adams & Nadol, *supra*, at Ex. 59, p. 8.) NF-kβ had previously been shown to be present in the cochlea in significant amounts and is believed to ordinarily have a protective effect in the inner ear. (*Id*. at 8-9.) However, it was also known to operate as a cellular stress pathway, producing cytokines when pathologically activated. (*Id*.) Prior studies demonstrated that noise exposure could activate NF-kβ within the cochlea. (Joe Adams, et al., *Selective Activation of NF-kB in the Cochlea by Sensory and Inflammatory Stress Running Head: NF-kB Activation*, 160(2) NEUROSCIENCE 530 (2009) (Ex. 66, p. 7).) A separate study also confirmed that damage to fibrocytes in the lateral wall of the cochlea, including from noise exposure, correlated to different degrees of hearing loss.[15] (Masuda & Kanzaki, *supra*, at Ex. 23, p. 6.) In 2009, Merchant and Adams published a study in which they were able to activate NF-kβ to cause injury within mouse cochlea by injecting liposaccharides to induce systemic inflammation. (Adams, et al., *supra*, at Ex. 66.) The result of this line of research is the "stress response theory," which purports to integrate the previously advanced hypotheses. (Masuda & Kanzaki, *supra*, at Ex. 23, p. 1.)

The stress response theory observes that the prior literature does not support a single local cause of SSNHL. Accordingly, the theory proposes in effect a final common pathway whereby broader factors can contribute to injury within the ear. (Masuda & Kanzaki, *supra*, at Ex. 23, p. 10.) The stress response theory suggests that stress leaves people susceptible to immune disruption both by reducing natural killer ("NK") cells, which help resist viral and bacterial infection, and by promoting production of proinflammatory cytokines IL-1 and IL-6. (*Id*. at 2-4.) The proinflammatory cytokine IL-6 is not limited to local influence via classical signaling but can act on the whole body by cleaving to IL-6 receptors from apoptotic neutrophils. IL-6 has been associated with activation of NF-kβ under stress. NF-kβ in turn can further induce IL-6 expression, creating a feedback loop. (*Id*. at 6.) Once this feedback loop occurs, activation of NF-kβ in the lateral wall of the cochlea disrupts homeostasis within the ear and results in damage to fibrocytes within the lateral wall leading to SSNHL. This process is depicted by the following chart discussed during the hearing:

[15] A "fibrocyte," also called a "fibroblast," is a flat, elongated connective tissue that forms fibrous tissue in the body. *Dorland's Illustrated Medical Dictionary*, p. 694 (33rd Ed. 2020). There are four types of fibrocyte within the lateral wall of the cochlea and the stria vascularis. (Masuda & Kanzaki, *supra*, at Ex. 23, p. 6.) Fibrocytes play a role in the process of maintaining homeostasis between sodium and potassium within the inner ear as discussed further below. (Masuda & Kanzaki, *supra*, at Ex. 23, p. 6.)



(Masatsugu Masuda, et al., *Correlations of Inflammatory Biomarkers With the Onset and Prognosis of Idiopathic Sudden Sensorineural Hearing Loss*, 33 OTOLOGY & NEUROTOLOGY 1142 (2012) (Ex. 68, p. 7).)

The stress response theory is viewed as a good candidate for explaining the clinical characteristics of idiopathic sudden hearing loss, including its unilateral presentation and prevalence in adulthood. (Masuda & Kanzaki, *supra*, at Ex. 23, p. 10.) Obviously, however, not everyone who experiences stress will lose their hearing. The stress response theory posits that the above-discussed process operates in connection with innate factors, such as polymorphisms or vascular tone, as well as possibly minor pre-existing subclinical damage to the inner ear. (*Id*. at 10.) Based on the specific findings of prior studies it does not appear to be the case that infection alone can cause sufficient NF-kβ activation to result in the type of fibrocyte damage that causes hearing loss (*Id*. at 9.) The process proposed by the stress response theory operates when multiple stressors combine synergistically to result in elevated IL-6. (*Id*. at 8.) This does not require a full clinical infection and may involve subclinical infection or other immune disturbance. (*Id*. at 9-10.)

Dr. Fife did not challenge the validity of the stress response theory. During the hearing, I specifically asked Dr. Fife to comment on the immunologic aspects of the theory. He declined, indicating that "I don't have any basis for challenging it." (Tr. 235-36.) Accordingly, I accept Dr. Hicks's reliance on the stress response theory as a sound and reliable explanation of the pathophysiology underlying SSNHL. Nonetheless, Dr. Fife challenges Dr. Hicks's causal opinion based on two peripheral concerns relating to whether the flu vaccine can be implicated by the stress response theory. First, Dr. Fife opines that an injection into the arm results in a localized reaction that does not reach the ear. Second, Dr. Fife opines that a literature review fails to provide any evidence of any association between the flu vaccine and SSNHL.

Dr. Fife's opinion that a vaccination into the arm would not have any effect on the inner workings of the ear is premised on two contentions. First, vaccination results in a

localized reaction such that neither the substance of the vaccination itself nor the immune reaction caused by the vaccination would circulate to the location of the blood-labyrinthine barrier. And, second, even if it did, the blood-labyrinthine barrier excludes most molecules from the inner ear. On this record, neither of these specific contentions persuasively rebuts Dr. Hicks's application of the stress response theory within the context of a post-influenza vaccination injury.

The stress response theory Dr. Hicks proposes does not actually contend that any bacterial or viral antigen "attacks" the inner ear. (Masuda & Kanzaki, *supra*, at Ex. 23, p. 10; Tr. 153-54.) Rather, as explained above, the theory proposes that an immune response outside the ear will lead to an increase in circulating cytokines that will have an inflammatory effect on the inner ear and ultimately lead to damage to the cochlear lateral wall via the NF-kβ pathway. Asked whether a cytokine response could enter the ear, Dr. Fife initially indicated that:

> [C]ertainly flu vaccine does trigger a cytokine response. That's the whole point of giving the vaccine . . . [b]ut that response is primarily localized to the site of the immunization . . . As far as a more systemic response or response in another part of the body like the inner ear, again, it's hard to know exactly how that would happen, because as I said, the primary response is in the site of the immunization and the regional lymph nodes there, but that tends not to be a systemic response.

(Tr. 213.)

Importantly, however, petitioner has filed literature supporting the idea that the flu vaccine can result in increased post-vaccination IL-6 circulating in the bloodstream as required by the stress response theory. A study filed by petitioner, Carty, et al., found that subjects receiving the flu vaccine experienced an inflammatory acute response phase that included a 14-40% increase in IL-6 found in blood samples taken at 24 hours post-vaccination.[16] (Cara Carty, et al., *Inflammatory Response After Influenza Vaccination in Men With and Without Carotid Artery Disease*, 26 ARTERIOSCLEROSIS, THROMBOSIS, AND VASCULAR BIOLOGY 2738 (2006) (Ex. 33, p. 3).) Notably, measurement at 24 hours is consistent with the stress response theory as that theory indicates that IL-6 expression and NF-kβ activation occur within 24 hours.[17] (Masuda &

[16] The study was designed to compare men with severe carotid artery disease ("CAAD") against men without stenosis. (Carty, et al., *supra*, at Ex. 33, p. 1.) However, the authors found that "[t]he postvaccination levels of all markers were significantly higher than baseline levels" for both men with CAAD and men without CAAD. (*Id*. at 3.) Specifically, there was a 40.1% increase in IL-6 among men with CAAD and a 14% increase in men without CAAD. The increase was characterized as "mild, but measurable." (*Id*. at 1 (conclusion).)

[17] Another study filed by petitioner contained a potentially confounding finding given that the stress response theory relies in part on a *reduction* in NK cells. Jost, et al., found that NK cells, and particularly CD56, were *increased* in blood samples drawn between 4-7 days following intramuscular vaccination (Stephanie Jost, et al., *Changes in Cytokine Levels and NK Cell Activation Associated with Influenza*, 6(9) PLOS ONE e24060 (2011) (Ex. 24, p. 3).) CD56 cells in turn "have a higher capacity to produce cytokines as well as a more important proliferative potential than other NK cell subsets." (*Id*. at 5.) This finding was

Kanzaki, *supra*, at Ex. 23, pp 6-7, Table 3.) Providing still greater consistency with the stress response theory, an additional study by Glaser, et al., demonstrated that subjects experiencing depressive symptoms (subjects were under stress as caregivers for family members with progressive dementia or as caregivers who recently lost their family member) produced an amplified inflammatory reaction to the flu vaccine in the form of increased levels of IL-6 compared to controls as measured at two-weeks post-vaccination.[18] (Ronald Glaser, et al., *Mild Depressive Symptoms Are Associated With Amplified and Prolonged Inflammatory Responses After Influenza Virus Vaccination in Older Adults*, 60 ARCHIVES OF GEN. PSYCHIATRY 1009 (2003) (Ex. 35).)

Dr. Fife ultimately conceded that cytokines could be released into the bloodstream from the site of vaccination but suggested that the concentration would be "extremely low" and "very diluted" based on the amount of circulation that reaches the inner ear. (Tr. 214-15.) In that regard, Dr. Hicks explained that the total quantity of fluid in the cochlea is only about two and a half drops. Blood supply to the inner ear represents only 1/1 millionth of the cardiac output of a human being. (Tr. 106, 109.) For these reasons, Dr. Hicks explained that homeostasis within the ear is extremely delicate with fluid exchange in the inner ear implicating only minute quantities. (*Id*.) Dr. Hicks indicated that the question is not quantity per se, but cytokine imbalance that affects the NF-kβ that is already present within the ear. (Tr. 156-57.) Nonetheless, Dr. Hicks also noted that the labyrinthine artery is considered a major artery. (Tr. 155-56.)

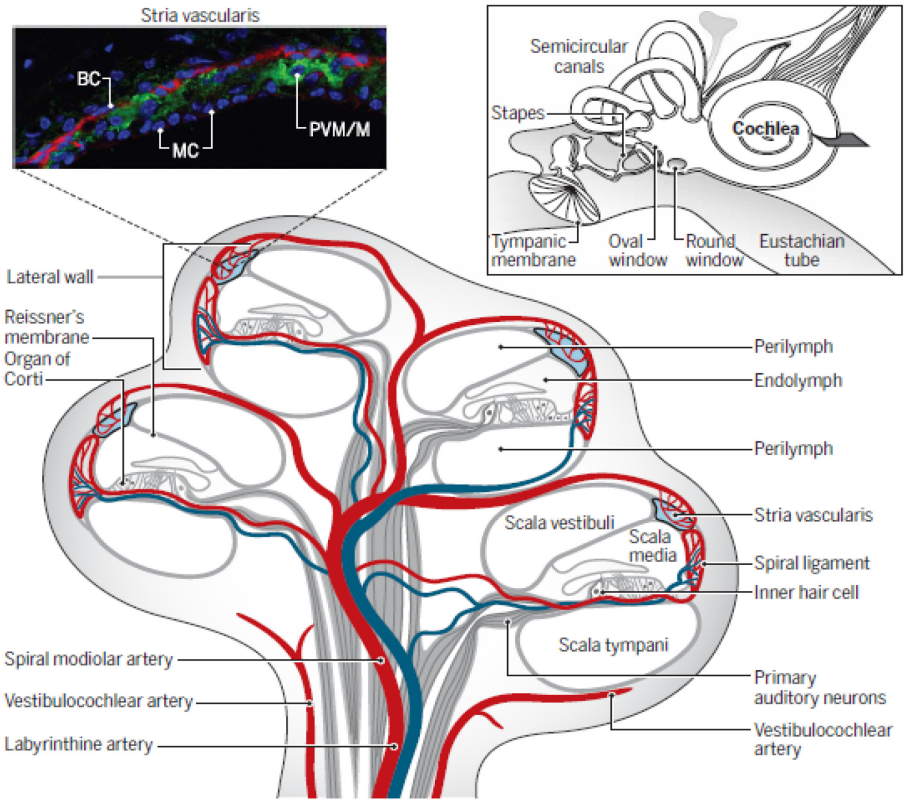
The stress response theory posits a "synergistic" effect whereby circulating cytokines converge with a stress-related abnormal immune response to trigger the above-discussed feedback loop. (Masuda & Kanzaki, *supra*, at Ex. 23, p. 10.) The theory does not require an active clinical infection, but may include subacute infection or other immunologic stressors. (*Id*. at 9-10.) No threshold degree of inflammation has been identified nor was Dr. Fife able to articulate the degree of dilution he was positing. (Tr. 214.) Additionally, to the extent Dr. Fife's opinion is premised more broadly on the small amount of circulating blood that reaches the inner ear, he did not explain how this opinion squares with his stated inability to challenge the immunology underlying the stress response theory more generally or with the fact that oral medications, which likewise reach the inner ear via the bloodstream, have been shown to have both ototoxic and therapeutic effects on the inner ear. Referring to ototoxic drugs, Dr. Hicks explained "[i]t gets there. I mean, the microcirculation of the inner ear is extremely small, but it's also extremely sensitive, and minor changes can lead to major problems." (Tr. 151-52.) In light of his otolaryngology specialty, Dr. Hicks is better positioned than Dr. Fife to speak to the specifics of the relationship between the circulatory system and the inner ear.

---

in contrast to subjects experiencing acute influenza infection, who saw a decrease in NK cells. Nonetheless, the vaccinated subjects also showed "very low and stable" cytokine levels, casting doubt on the specific effect of the observed increased NK cells. (*Id*. at 4.) Notably, IL-6 was not measured. (*Id*. at 3.)

[18] This study only included measurement of IL-6 at two weeks post vaccination. Accordingly, this study does not shed light on how quickly the IL-6 increased during the immediate post-vaccination period.

This also leads to Dr. Fife's second premise, which is that the blood-labyrinthine barrier, like the blood-brain barrier, tends to exclude proteins such as cytokines. (*Id*.) This point again places the different specialties of the two experts (otolaryngology versus infectious disease) in sharp relief. During the hearing, Dr. Hicks devoted significant time to explaining how the blood-labyrinthine barrier works. (Tr. 106-16.) He explained that the idea that the inner ear is immunologically privileged as a result of the blood-labyrinthine barrier is outdated. (Tr. 102.) In fact, the idea of an immunologically privileged inner ear has been under question since the 1980's. (Solares, Hughes & Tuohy, *supra*, at Ex. 69, p. 2.) Reviewing a graphic contained in an article filed by respondent (Nyberg, et al., Ex. I, *infra*), Dr. Hicks explained that the stria vascularis, which represents the boundary between inner and outer ear, is weak and susceptible to inflammatory proteins. (Tr. 103-04.) Dr. Hicks explained the below image in depth during the hearing:



(Sophie Nyberg, et al., *Delivery of therapeutics to the inner ear: The challenge of the blood-labyrinth barrier*, 11 SCI. TRANSLATIONAL MED. eaao0935 (2019) (Ex. I, p. 3).)

This image is a cross section of the cochlea. It demonstrates that the labyrinthine artery enters the cochlea and branches down to the capillary level at the scala media. The scala media is the central portion of the cochlea and is surrounded by the scala vestibuli and scala tympani. The scala media is high in potassium while the surrounding scala tympani and scala vestibuli contain perilymph that is high in sodium. The stria vascularis (depicted on the far right of the diagram) provides blood supply to the scala media and maintains homeostasis by balancing the potassium content of the scala

media and the sodium in the perilymph. (Tr. 106-07.) This exchange generates the electrical energy that sends sound to the brain. (Tr. 114.)

The stria vascularis is perfused by systemic blood circulation. (Tr. 109.) The "cobweb" of capillaries in the stria vascularis, and the extent to which they will or will not allow for fluid exchange, are what constitutes the blood-labyrinthine barrier. (Tr. 114.) The stria vascularis contains "nonfenestrated" capillaries. (Nyberg, et al., *supra*, at Ex. I, p. 4.) The barrier consists of "tight junctions" that decrease the rate of entry into the perilymph from the blood by compounds of increasing molecular weight. (*Id*.) However, the vasculature of the blood-labyrinthine barrier is subject to degeneration caused by age, metabolic changes, noise damage, or inflammation, all of which can increase permeability. (*Id*. at 2-3.) According to Dr. Hicks, inflammation dilates the blood vessels within the stria vascularis and facilitates influx into the fluid contents of the inner ear. (Tr. 109-12.) Dr. Hicks indicated that this is how orally administered otoprotective therapeutics work as well as how orally administered ototoxic drugs have their damaging effect. (Tr. 109-13.)

Relying on Nyberg, et al., however, Dr. Fife, stressed that the blood-labyrinthine barrier inhibits drug delivery to the inner ear, making it less likely that something like a vaccine antigen could cross that barrier. (Nyberg, et al., *supra*, at Ex. I; Tr. 215-16.) Dr. Fife indicated, by way of analogy, that the blood-brain barrier allows steroid treatment through but still offers a barrier to other substances, including cytokines. (Tr. 214, 224.) Nyberg, et al., indicate, however, that the blood-labyrinthine barrier is not analogous to the blood-brain barrier, but rather is considered "physiologically more complex" than the blood brain barrier. (Nyberg, et al., *supra*, at Ex. I, p. 2.) Moreover, on cross-examination, Dr. Fife conceded that the Nyberg article confirms that the blood-labyrinthine barrier is permeable to molecules and that inflammation can modulate the permeability of the blood-labyrinthine barrier as petitioner posits. (Tr. 226-27.) In fact, Nyberg, et al., specifically hypothesize that the microvasculature system of the stria vascularis regulates blood flow and permeability via inflammatory cytokines. (Nyberg, et al., *supra*, at Ex. I, p. 5.) Additionally, Solares, et al., filed by petitioner, concluded in the context of bilateral autoimmune sensorineural hearing loss that signaling molecules, such as cytokines, can traverse the blood labyrinthine barrier and activate autoreactive T-cells within the inner ear. (Solares, Hughes & Tuohy, *supra*, at Ex. 69; Tr. 103 (Dr. Hicks); Tr. 226 (Dr. Fife).)

Finally, Dr. Fife's opinion was also based in significant part on his literature review which indicated to him that SSNHL is generally idiopathic and that there is no established association between the flu vaccine and SSNHL. (Tr. 208-09.) In that regard, respondent has come forward with two studies seeking to address that causal association. However, especially in light of petitioner's above-discussed demonstration of the stress response theory as a sound and reliable concept underlying Dr. Hicks's causal opinion, neither study is adequate to cast significant doubt on the reliability of Dr. Hicks's opinion. Petitioner is not obligated to present an epidemiological case supporting his claim. *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317,

1325 (Fed. Cir. 2006). Moreover, there are reasons to doubt the significance of the findings presented in these two studies.

First, Williams et al., conducted a study in 2011 of 212 VAERS reports related to neurological conditions following administration of the H1N1 influenza vaccination. (Ex. D.) Although this study did not capture SSNHL as a neurological injury following the H1N1 vaccination, this study was limited to "serious" conditions, which it defined as reports of hospitalization, prolongation of hospitalization, permanent disability, or conditions considered life-threatening. (*Id*. at 3 (methods).) SSNHL can be permanent but is also considered reversible with prompt care and has a variable prognosis. (Ex. 50, p. 2.) It is estimated that between 32-65% of SSNHL cases recover spontaneously. (Stachler, et al., *supra*, at Ex. 55, p. 4.) Accordingly, it is not clear what proportion of SSNHL cases this study was equipped to detect.

Second, Baxter, et al., conducted a case-centered analysis of SSNHL following vaccination by reviewing the Kaiser Permanente Northern California database for a period from 2007 to 2013. (Ex. F.) During the review period, over 20 million vaccines overall, and over 8 million flu vaccines specifically, were administered. Notably, the odds ratio did detect an increased likelihood that a person received a flu vaccine within 1-14 days or 1-28 days prior to the onset of their SSNHL (*Id*. at 5 (Table 2)); however, the authors rejected any association because the confidence interval suggested the results were not statistically significant (*Id*. at 4). The authors noted that a limitation of their study is that only medically attended cases of SSNHL would have been captured. Additionally, those cases that were included were not individually reviewed to confirm diagnosis and/or onset date. (*Id*. at 6.) Also significant, the authors considered the date of onset to be the date on which any hearing-related diagnosis was given. (*Id*. at 3.) Dr. Hicks explained, however, that delay in treatment is a major problem in the clinical care of SSNHL. (Tr. 176-79; *see also* Massachusetts Eye and Ear, *Diseases and Conditions: Sudden Deafness* (Ex. 39, p. 2) (noting that delay in diagnosis is the "greatest clinical problem in SSNHL.") Because SSNHL symptoms can be confused with benign issues like wax impaction or congestion, patients often wait days or weeks before seeking medical evaluation and then may be treated symptomatically for a period before being referred for diagnosis. (Massachusetts Eye and Ear, *supra*, at Ex. 39, p. 2.) Thus, in light of Dr. Hicks's testimony, these study limitations appear to be especially meaningful to the identification of cases of SSNHL temporally proximate to flu vaccination. This suggests a need to interpret the study cautiously, particularly given that the study included an equivocal finding of an association even with these limitations.

For all of the reasons discussed above, petitioner has met his burden of proof with respect to *Althen* prong one by presenting a sound and reliable explanation through Dr. Hicks of how a flu vaccine can cause SSNHL via the stress response theory. Two prior decisions provide detailed analysis of whether the flu vaccine can cause SSNHL. *Doe/16 v. Sec'y of Health & Human Servs.*, No. 06-670V, 2008 WL 2390064 (Fed. Cl. Spec. Mstr. June 2, 2008); *Inamdar v. Sec'y of Health & Human Servs.*, No. 15-1173V, 2019 WL 1160341 (Fed. Cl. Spec. Mstr. Feb. 8, 2019). Both decisions concluded that the petitioner had not met their burden; however, neither of these prior cases addressed

the specific stress response theory advanced by petitioner in this case, though the petitioners in those cases did rely on the presence of post-vaccination inflammation seemingly without an explanation (such as the stress response theory) for how they could affect the inner ear and/or be injurious.

Petitioner also advanced a theory of reactivation of a latent HSV-1 infection. In fact, Dr. Hicks hypothesized a role for viral reactivation within the context of the stress response theory. (Ex. 50, p. 8.) However, Dr. Hicks explained that his proposed theories could operate alone as well as in combination. (Ex. 50, p. 8 (opining injury was due to "one or more of the following medical theories.") Because I have accepted Dr. Hicks's reliance on the stress response theory in itself, I do not reach the question of whether petitioner's HSV-1 reactivation theory satisfies his burden of proof under *Althen* prong one.

b. *Althen* Prong Two

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show [s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. *See* Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing ... that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted").

There is no dispute in this case that petitioner's post-vaccination presentation was properly diagnosed as SSNHL. (Tr. 95 (Dr. Hicks), 208-09 (Dr. Fife).) Additionally, petitioner presents the opinions of two physicians with whom he has a treatment relationship, both of whom opine that petitioner's flu vaccine caused his SSNHL. Dr. Erner, a doctor of osteopathic medicine who previously saw petitioner prior to vaccination for reduced energy and general malaise, submitted a report opining that petitioner's flu vaccine combined with his concurrent bacterial folliculitis to cause his SSNHL. (Ex. 14, p. 2.) Dr. Nicora, a doctor of internal medicine and petitioner's primary care physician from April of 2012 forward,[19] testified that he felt comfortable

[19] This is notable in that Dr. Nicora did not have a doctor-patient relationship with petitioner either before or immediately after onset of his SSNHL. *See, e.g. Nuttall v. Sec'y of Health & Human Servs.*, 122 Fed. Cl. 821, 832-33 (2015) (noting that the opinions of treating physicians are not necessarily entitled to

ruling out doxycycline as a cause of petitioner's SSNHL on the basis that it is not ototoxic and therefore concluded that systemic inflammatory effects of the flu vaccine were a more likely cause of SSNHL in petitioner's case. (Tr. 20-23.) Dr. Nicora based his assessment on the inflammatory properties of vaccination, the temporal association between the vaccination and petitioner's hearing loss, the absence of any other insulting agent at the time, and the absence of any other potential cause. (Tr. 25.) Petitioner's medical records also confirm that following complete work up, insidious causes of hearing loss were ruled out. (Ex. 10, p. 3.) Of course, neither Dr. Erner nor Dr. Nicora are specialists in either otolaryngology or infectious disease; however, petitioner's testifying otolaryngology expert, Dr. Hicks, also opined that vascular occlusion or ischemia and intra-labyrinthine membrane break do not explain petitioner's SSNHL. He also opined that petitioner's flu vaccine caused petitioner's SSNHL via the stress response theory. (Ex. 50, pp. 7-8.)

As explained above, Dr. Fife did not challenge the validity of the stress response theory as an explanation for the etiology of SSNHL generally, but questioned Dr. Hicks's specific application of that theory to vaccination. However, Dr. Fife did acknowledge that SSNHL is an acquired condition. (Tr. 222.) He also acknowledged that some triggering events for SSNHL are immune-mediated and that corticosteroids are a primary treatment for SSNHL because it is suspected to have an inflammatory cause. (Tr. 223.) Dr. Fife agreed that petitioner had no prior hearing loss and suffered a distinct change in his hearing on December 12, 2011. (Tr. 217.) Dr. Fife also confirmed that he is not proposing any alternative etiology for petitioner's condition. (Tr. 222-23.) Notwithstanding his ultimate disagreement as to vaccine-causation, these points are consistent with a logical sequence of cause and effect between petitioner's SSNHL and an antecedent (albeit unknown) immune and/or inflammatory trigger.

Inner ear disorders are by their nature very difficult to investigate because the inner ear cannot be penetrated in an ordinary clinical setting. (Ex. 50, pp. 6, 9.) Moreover, Dr. Hicks stressed the delicacy of the inner ear to minute changes and did not suggest that the stress response theory would lead to outward clinical signs of inflammation beyond the manifestations of SSNHL itself. For example, Dr. Hicks indicated that lab results are generally not beneficial in assessing SSNHL. (Ex.50, p. 2.) Instead, diagnosis is usually based on history, examination, and MRI, with 90 percent of the diagnosis being the history. (Ex. 50, p. 2; Tr. 87.) Accordingly, further clinical findings beyond the fact of petitioner having otherwise unexplained sudden hearing loss temporally proximate to his vaccination are not necessarily expected. As noted above, 85-90% of SSNHL cases remain idiopathic despite investigation in the course of care.

However, one additional clue does potentially exist in petitioner's lab results. When petitioner presented for treatment four days post-vaccination, he had bloodwork drawn. As of December 13, 2011, his neutrophil count was elevated. (Ex. 3, p. 22.) According to a 2012 study by Masuda, et al., abnormal neutrophil counts were correlated in SSNHL subjects not only to prognosis, but also to both elevated IL-6 and

---

added weight when they saw the petitioner only after the injury at issue), *aff'd* 640 Fed.Appx. 996 (Fed. Cir. 2016).

decreased NK cells, both of which are expected under the stress response theory. (Masuda, et al., *supra*, at Ex. 68.) Dr. Fife opined that petitioner's elevated neutrophils are not clinically significant because they occur in the context of a normal finding for total white blood cells. Additionally, the finding may be attributable to the previously diagnosed folliculitis. (Tr. 211.) For these reasons the finding could not in itself be dispositive. However, for purposes of their study, Masuda, et al., considered any neutrophil finding above the reference range to be abnormal and concluded that "[n]eutrophil counts above the reference range of a facility *will be a useful indicator* for poor prognosis of ISHL." (Masuda, et al., *supra*, at Ex. 68, p. 8 (emphasis added).) In fact, the authors also hypothesized that increased neutrophil counts might be sufficient to activate the stress response in the cochlear lateral wall independent of IL-6. (*Id*. at 7.) Even if petitioner's lab work does not provide strong or definitive evidence supporting causation, it is still significant that the lab results are consistent with petitioner's theory. Dr. Hicks primarily focused on the neutrophil finding as relating to prognosis but also confirmed that it supports this theory of causation. (Tr. 170, 185-86.)

Petitioner's clinical history is also significant for additional sources of stress that could hypothetically contribute to the synergistic effect anticipated by the stress response theory. *Accord Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). First, petitioner's medical records suggest some degree of chronic stress and/or depression during the years and months leading up to his hearing loss. Specifically, petitioner raised the issue of stress with Dr. Wein in both 2010 and 2011. (Ex. 1, pp. 6, 28.) By September 25, 2011, petitioner was being referred for treatment for symptoms of reduced energy and general malaise, though no diagnosis or resolution of these complaints is reflected in the medical records. (Ex. 49, p. 84.) More acutely, Dr. Erner suggested that petitioner's folliculitis may have contributed an increased inflammatory state. (Ex. 14, p. 2.) However, none of these factors have been cited as constituting any independent alternative cause of petitioner's SSNHL.

Dr. Hicks also combined his discussion of the stress response theory with the additional idea that petitioner's flu vaccine may have reactivated a latent HSV-1 infection that contributed to the inflammatory conditions within the ear. (Ex. 50, p. 8.) HSV-1 infection has been separately associated with SSNHL and Dr. Hicks opined that it can be reactivated by innocuous genotoxic stressors or infection. (*Id*.) As noted above, I did not find it necessary to reach that aspect of Dr. Hicks's opinion. I do note, however, that if one were to conclude Dr. Hicks's opinion was premised on the presence of a latent HSV-1 infection, petitioner did testify to having a history of cold sore presentation. (Tr. 36-38.) Respondent's expert, Dr. Fife, additionally testified that he agreed petitioner's history indicates a latent HSV-1 infection, characterizing the history as "pretty typical." (Tr. 228.) To the extent this also raises the question of whether HSV-1 reactivation alone could have caused petitioner's SSNHL, none of the treating physicians or experts in this case so opined. Additionally, all three testifying physicians indicated that they could not conclude that petitioner's folliculitis, which predated petitioner's flu vaccination, itself constituted a manifestation of HSV-1 infection. (Tr. 32-33, 172, 213.) Dr. Hicks specifically opined that petitioner's folliculitis

alone would not have been the cause of petitioner's SSNHL under his stress response theory. (Tr. 173.) Dr. Fife did not opine on that point.

In light of all of the above, petitioner has met his burden of proof with respect to *Althen* prong two.

c. *Althen* Prong Three

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 Fed. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Human Servs.*, No. 11-355V, 2013 WL 3214877 at *26 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

During the hearing, Dr. Fife explained that the type of cytokine response proposed in this case develops rapidly, over the course of days, if not hours. (Tr. 218-19.) Similarly, the literature filed by petitioner indicates that under the stress response theory NF-kβ activation within the cochlea likely occurs within 24 hours of a loading stressor. (Masuda & Kanzaki, *supra*, at Ex. 23, p. 6.) Dr. Hicks similarly testified that activation of NF-kβ would occur within 24-36 hours of vaccination. (Tr. 187-88.) Subsequent to that process, SSNHL is by definition a change in hearing that then occurs over the course of up to 72 hours. (Stachler, et al., *supra*, at Ex. 55, p. 4.) Accordingly, pursuant to petitioner's theory, hearing loss should be evident by no later than four days post vaccination.

Petitioner received his flu vaccination on December 9, 2011, which was a Friday. (Ex. 1, p. 3.) He was first seen for complaints that included ringing in his left ear on Tuesday, December 13, 2011, four days post-vaccination. (Ex. 3, p. 4.) At that time, he reported a history that placed onset of his symptoms the day prior, on Monday, December 12, 2011, which is three days post vaccination. (*Id.*) Loss of hearing was not specifically included in that history. (*Id.*) However, during the entitlement hearing petitioner described onset of auditory symptoms ("the sound was weird") and aural fullness beginning the Monday following his vaccination. (Tr. 44-45.) Dr. Hicks confirmed that tinnitus and aural fullness can be an initial symptom of SSNHL. (Ex. 50, p. 3.) In any event, although not included within the reported history, petitioner's December 13, 2011 assessment confirmed that petitioner was experiencing a decrease in hearing as of that time. (*Id.* at 6.) Accordingly, there is preponderant evidence that

petitioner suffered SSNHL by no later than four days post-vaccination, timing which is consistent with petitioner's reliance on the stress response theory.

In light of the above, petitioner has satisfied his burden of proof under *Althen* prong three.

d. Factor(s) unrelated to vaccination

Once petitioner has satisfied his own burden of proof pursuant to the *Althen* test, the burden shifts to respondent to demonstrate that the injury was caused by factors unrelated to vaccination. § 300aa-13(a)(1)(B); *Deribeaux v. Sec'y of Health & Human Servs.*, 717 F.3d 1363, 1367 (Fed. Cir. 2013). As noted above, respondent's expert, Dr. Fife, confirmed that he has not proposed an alternative etiology for petitioner's hearing loss. (Tr. 223.) Additionally, petitioner's medical records confirm that insidious causes of hearing loss were ruled out. (Ex. 10, p. 3.) Accordingly, respondent has not presented preponderant evidence that petitioner's hearing loss was caused by any factor other than vaccination.

VI. Conclusion

Accordingly, for all the reasons described above, I find that petitioner is entitled to compensation. Specifically, I find that petitioner has established by preponderant evidence that his SSNHL was caused-in-fact by his December 9, 2011 influenza vaccination. A separate damages order will be issued.

IT IS SO ORDERED.

s/Daniel T. Horner
Daniel T. Horner
Special Master